Paper

INTER-SPECIES EXTRAPOLATION OF SKIN HEATING RESULTING FROM MILLIMETER WAVE IRRADIATION: MODELING AND EXPERIMENTAL RESULTS

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Abstract—**This study reports measurements of the skin surface temperature elevations during localized irradiation (94 GHz) of three species: rat (irradiated on lower abdomen), rhesus monkey (posterior forelimb), and human (posterior forearm). Two exposure conditions were examined: prolonged, low power density microwaves (LPM) and short-term, high power density microwaves (HPM). Temperature histories were compared with calculations from a bio-heat transfer model. The mean peak surface temperature increase was approximately 7.0°C for the short-term HPM exposures for all three species/locations, and 8.5°C (monkey, human) to 10.5°C (rat) for the longer-duration LPM exposures. The HPM temperature histories are in close agreement with a one-dimensional conduction heat transfer model with negligible blood flow. The LPM temperature histories were compared with calculations from the bio-heat model, evaluated for various (constant) blood flow rates. Results suggest a variable blood flow model, reflecting a dynamic thermoregulatory response, may be more suited to describing skin surface temperature response under long-duration MMW irradiation.**

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Key words: radiation, non-ionizing; skin dose; modeling, dose assessment; radiation damage

INTRODUCTION

THE MILLIMETER wave band (MMW) is a subset of the radio frequency (RF) portion of the electromagnetic spectrum, comprising the range 30–300 GHz. Devices using MMW technologies include automobile collision avoidance systems (Moffa et al. 1996; Gilbert et al. 1997), medical and dental applications (Nikawa et al. 2000; Carl et al. 2000), wireless communications systems (Pakhomov et al. 1998; Ryan et al. 2000) and military devices (Ryan et al. 2000). The increasing use of RF signals in the MMW band in both defense and civilian applications necessitates a better understanding of the bioeffects within this frequency range.

The primary effect of MMW exposure is surface heating (Durney et al. 1986). The power density threshold for human perception of skin heating is lower for MMW irradiation than for longer-wavelength exposures in the RF spectrum (Blick et al. 1997). It is not clear whether MMW irradiation produces a greater skin temperature increase (for a given power density) than occurs at longer wavelengths.

Millimeter wave irradiation is absorbed in the superficial tissues. The tissue penetration depth is approximately 0.04 cm at 94 GHz, compared with a penetration depth of 3.2 cm at 2.45 GHz (Blick et al. 1997). The superficial absorption characteristics mean exposure standards must be based on power density rather than on the normalized specific absorption rate (SAR) (IEEE 1999).

The local temperature rise may be affected by heat conduction to surrounding tissue, advection (blood flow), surface convection (Foster et al. 1978) and other thermoregulatory responses (e.g., sweating). These factors can profoundly alter surface temperatures for a given exposure scenario, making it difficult to predict thermal responses on the basis of power density alone.

The use of animal models for evaluation of skin heating effects from MMW irradiation is controversial, as there may be significant difficulties with extrapolating the results to humans. Differences in tissue composition, skin blood flow, and surface cooling mechanisms may complicate the interpretation of results obtained from animals and their application to humans. For example, sweat evaporation is a major thermoregulatory mechanism that limits heat gain in humans. Rodents do not sweat, although rats may employ evaporative surface

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cooling by licking the fur. The rhesus monkey (*Macaca mulatta*) and patas monkey (*Erythrocebus patas*) employ eccrine sweating for heat dissipation (Johnson and Elizondo 1979; Kolka and Elizondo 1983), while the squirrel monkey (*Saimiri sciureus*) exhibits sweating only on the hands and feet (Stitt and Hardy 1971). However, no commonly-used laboratory animal sweats as efficiently as do humans.

While humans have an active vasodilation mechanism to increase skin blood flow during heating, other species do not dilate the cutaneous vascular bed, except at some limited anatomical sites (e.g., the rabbit ear, the rat tail).

Walters et al. (2000) developed a one-dimensional conduction model of MMW heating of the skin surface. That model explicitly neglects thermal advection (blood flow) and may be appropriate to describing short-term exposure in which there is neither sufficient time for active thermoregulatory response (sweating, vasodilation), nor time for significant thermal diffusion from the exposed region.

The present investigation sought to (1) identify conditions under which animal models may be used to predict surface temperature increases resulting from MMW irradiation in humans, and (2) determine whether a simple, closed-form model incorporating blood flow could describe superficial heating from MMW irradiation.

The model presented here is based on a closed-form solution to the modified, one-dimensional Bio-Heat Transfer Equation.

METHODS

Far-field exposures were performed on four subjects for each of the three species considered (rat, rhesus monkey, human). The exposure system employed for the human subjects is illustrated in Fig. 1. Exposures of rhesus monkeys and rats were performed with the same system, with the anesthetized animals positioned on the elevated platform used for the human arm support (Fig. 1).

The animals involved in this study were procured, maintained, and used in accordance with the Federal Animal Welfare Act and the "Guide for the Care and Use of Laboratory Animals," prepared by the Institute of Laboratory Animal Resources—National Research Council. All animal experiments and animal care procedures were approved by the Institutional Animal Care and Use Committee of the Air Force Research Laboratory, Brooks Air Force Base.

The use of human subjects in this research was in accordance with a protocol approved by Institutional Review Board (Brooks Air Force Base, Air Force Laboratory Advisory Committee on Human Experimentation), and by the Office of the Surgeon General of the Air Force.

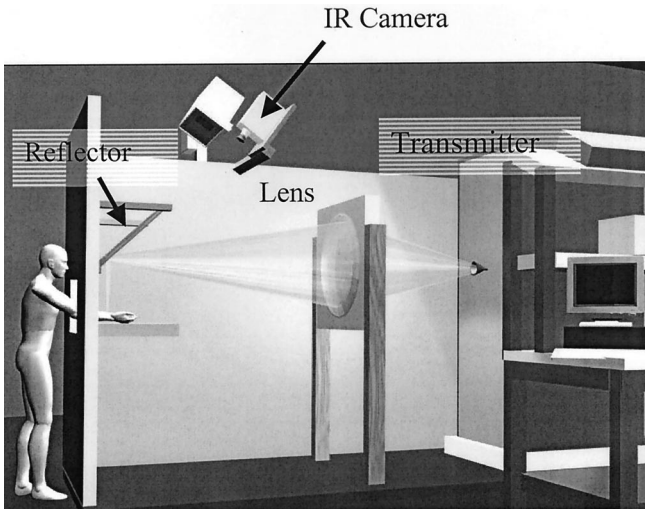

Fig. 1. The experimental system is illustrated, as configured for exposures of the human forearm. The same system was used for animal exposures, with the subject animal positioned on the platform. Human and monkey exposures were performed on the posterior of the forelimb.

MMW exposure system

A 50-W transmitter manufactured by Applied Electromagnetics, Inc. (Philadelphia, MS), with a Varian VKB2462L2 gridded extended oscillator emitted microwaves at a frequency of 94 GHz. A gaussian dielectric lens (Shelton 1991) was used to augment the maximal available power density by focusing the output of the transmitter into a beam with a diameter of 3.3 cm. The input side of the lens had a focal length of 90 cm, so the lens was positioned 90 cm from the conical output antenna of the transmitter. Varying the duty factor of a 1,000 pulse per second rectangular wave train that gated the output of the transmitter produced steps in power density below the maximum. A computer program calculated pulse durations necessary to produce the required duty factors (50–90%). A programmable oscillator generated pulse trains with the required duty factors and durations.

Two different exposure scenarios were tested on each subject: a sustained, low incident power microwave (LPM) exposure (175 mW cm^{-2} for 180 s) and a short-term, high incident power (HPM) exposure (1.0 W cm^{-2} for 3.0 s). A representative image of the irradiated region on the human forearm is shown in Fig. 2. The area of uniform irradiation was approximately 1 cm in diameter, as judged by the rate of temperature increase (temperature increase of 90–100% of maximum).

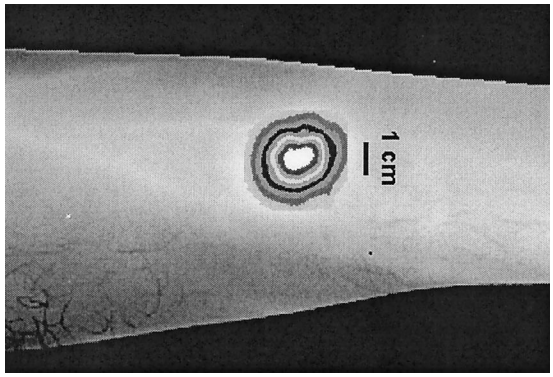

Fig. 2. The irradiated area of the posterior surface of the human forearm is shown, with isotherms determined by IR thermography.

Each subject was exposed three times at each power density, with sufficient intervals between exposures to allow thermal re-equilibration. Rats were irradiated on the right abdomen; humans were irradiated on the posterior and anterior surfaces of the forearm; monkeys were irradiated only on the posterior surface of the forelimb. All subjects were shaved over the irradiated areas.

Temperature measurement

Skin surface temperatures were measured continuously before, during, and immediately after exposure, using a Radiance I infrared camera system (Amber Engineering, Inc., Goleta, CA). The camera contains a 256 × 256 focal plane array of indium antimonide sensors. With the region of interest located 1.0 m from the camera, each sensor measured the temperature of a patch of skin 500 μm × 500 μm. Images were sampled at a rate of 10 s^{-1} for the HPM exposures and 0.2 s^{-1} for the LPM exposures. Automated image analysis was performed off-line using a LabView (National Instruments, Austin, TX) based program. Multi-point calibration with a Mikron M340 (Mikron Infrared, Inc., Oakland, NJ) black-body source provided a measured accuracy of ±0.1°C over the temperature range examined.

Subjects—rats

Four-mo-old male Sprague-Dawley rats were obtained from the colonies of Charles River Laboratories (Wilmington, MA). They were individually housed in standard plastic solid-bottom cages (26 × 23 × 20.5 cm) with water available ad libitum. Rats were kept on a food-restricted diet (Formulab 5008, Purina Meals, Inc., St. Louis, MO) to maintain weights within the range 380–400 g. All rats were weighed twice weekly.

Subjects—monkeys

Female rhesus monkeys (*Macaca mulatta*) were housed individually in stainless steel cages and provided a diet of monkey chow (Purina) supplemented with fresh fruit twice daily. Water was available ad libitum. Monkeys were 4 to 5 y of age and weighed 4–5 kg at the time of the experiments.

Subjects—humans. The human subjects were Caucasian volunteers (three male, one female). Subjects were military personnel, Department of Defense civilians, or contractors involved in research on biological effects of RF radiation. All human subjects were unpaid volunteers who read and signed an informed consent document prior to participation.

MATHEMATICAL MODEL

Temperature calculations were based on a form of the Pennes bio-heat transfer equation (Pennes 1948):

$$k_t \nabla^2 T + \dot{m}_b C_b (T_{art} - T) + \dot{q}_m = \rho_t C_t \frac{\partial T}{\partial t} \qquad (1)$$

where k_t is the thermal conductivity of the tissue, $\dot{m}_b$ is the blood flow rate per unit tissue volume, C_b is the specific heat of the blood, T_{art} is the arterial temperature, $\dot{q}_m$ is the metabolic heat production per unit volume, and ρ_t and C_t are the mass density and the specific heat of the tissue. The metabolic heating term $\dot{q}_m$ will be assumed negligible for the skin.

The RF heating term is not included in the general bio-heat equation (eqn 1) and must be added to the energy balance. For plane-wave irradiation incident on a planar, lossy dielectric surface, the energy penetration depth is a function of wavelength and the tissue permittivity (Durney et al. 1986). The energy deposition rate, per unit surface area, in tissue subject to MMW irradiation is described by (Walters et al. 2000):

$$q = \beta I_0 e^{-2x/L} \qquad (2)$$

where I_0 is the incident energy density (W cm^{-2}) and β is the energy absorption coefficient for human skin. At 100 GHz, the absorption coefficient $\beta = 0.67$ (Gandhi and Riazi 1986). The spatial variable x is the distance from the [planar] tissue surface and the parameter L is the energy penetration depth, defined as the depth at which the power density (Poynting vector magnitude) has decayed to e^{-2} of its value at the surface. At a frequency of 94 GHz, the energy penetration depth is approximately 4.0×10^{-2} cm (Durney et al. 1986).

The RF heating term (i.e., the right hand side of eqn 2) is divided by $2L$ (the depth of the heated tissue volume) and inserted into eqn 1. Dropping the metabolic heating term and rearranging the resulting equation

yields the energy balance

$$\rho_t C_t \frac{\partial T}{\partial t} = k_t \nabla^2 T + \frac{\beta I_0}{2L} e^{-2x/L} - \dot{m}_b C_b (T - T_{art}). \quad (3)$$

The energy balance (eqn 3) is non-dimensionalized by defining a dimensionless temperature $\hat{T}(x,t) = \theta^{-1}[T(x,t) - T_{art}]$ where $\theta = T_0 - T_{art}$, T_0 is the initial skin temperature and T_{art} is the arterial temperature. The dimensionless spatial variable is defined by $\hat{x} = L^{-1} \times x$. Dimensionless time is defined as $\hat{t} = \alpha L^{-2} t$, where α is the thermal diffusivity of the skin.

Tissue temperature is assumed a function of one spatial dimension (skin depth, x) and time. The resulting dimensionless equation is

$$\frac{\partial \hat{T}}{\partial \hat{t}} = \frac{\partial^2 \hat{T}}{\partial \hat{x}^2} + \left(\frac{\beta I_0 L}{k_t \theta}\right) e^{-2\hat{x}} - \left(\frac{\dot{m} C_b L^2}{k_t}\right) \hat{T}. \quad (4)$$

The dermal tissue is assumed homogeneous and isotropic. The skin surface is treated as adiabatic, except for the incident MMW irradiation (Foster et al. 1978). The solution to eqn (4) is obtained by the Laplace transform method (Arpaci 1966), yielding an expression for $\hat{T}(\hat{x},\hat{t})$. Evaluating that expression for $\hat{x} = 0$ yields a function for the skin surface temperature:

$$\hat{T}(0, \hat{t}) = \frac{\sigma}{4}[2\Omega^{-0.5} \times \mathrm{erf}(\Omega\hat{t})^{0.5} + e^{4\hat{t}} \times \mathrm{erfc}(2\hat{t}^{0.5}) - 1] + \exp(-\Omega\hat{t}) \quad (5)$$

where $\Omega = \dot{m}_b c_b L^2 k_t^{-1}$ is a dimensionless number representing the ratio of thermal advection (associated with skin blood flow) to conduction through the epidermal and dermal tissues. The quantity $\sigma = \beta I_0 L k_t^{-1}\ \theta^{-1}$ represents the ratio of the absorbed power to the nominal thermal conduction rate. The functions erf(x) and erfc(x) are the standard error function and the complementary error function, respectively.**

In situations where skin blood flow is negligible (i.e., $\Omega \approx 0$) the problem reduces to a simple conduction model. This yields a result similar to that of Walters et al. (2000).

RESULTS

High-power exposures

The experimental results of the high-power exposures (3.0 s at 1.0 W cm^{-2}) are presented as Fig. 3. Mean temperature increases (from the pre-exposure surface temperature for a given subject and experiment) are plotted for each species. Human forelimb results are shown for the posterior surface exposures only. Exposure of the anterior surface resulted in nearly identical temperature increases as did exposure of posterior surface.

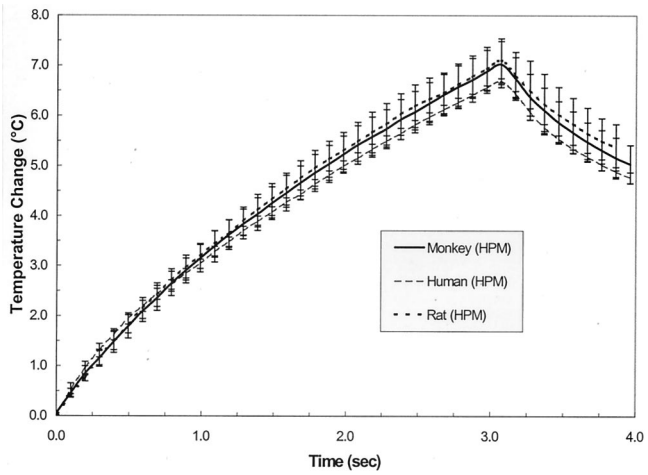

Fig. 3. The mean temperature increases are shown for 3-s HPM exposures of the three species. Four subjects per species were exposed with three repeated exposures per subject. Error bars represent ±1 S.D. of the species ensemble.

There is remarkable similarity in the responses of the three species to high-power exposures, with the peak mean temperature increase for the human subjects slightly lower than the corresponding mean values for the rats and monkeys.

The temperature increase data were fit to eqn (5) under conditions of no blood flow ($\Omega = 0$). The value of the parameter $\sigma = -3.01$ represents the best fit to the rat temperature increase data. The resulting function, and the mean temperature increases are shown as Fig. 4. Note this assumes blood flow is thermally insignificant for the short-term, high-power exposures.

The data of Fig. 4 are presented with the time scale non-dimensionalized by the characteristic diffusion time $\tau = L^2\alpha^{-1}$. This represents an estimate of the time scale for thermal conduction to occur over the distance L. Setting $L = 4.0 \times 10^{-2}$ cm and $\alpha = 1.0 \times 10^{-3}$ cm^2 s^{-1} (Mitchell et al. 1970) yields a diffusion time scale $\tau =$ 1.6 s. For purposes of non-dimensionalization, the thermal diffusivity α, and microwave penetration depth L were assigned the same respective values for each of the three species considered.

This is only a very crude estimate of the diffusion time scale, as there is large variability in the reported values for the thermal diffusivity of skin. Werner et al. (1992) report a thermal diffusivity of human epidermis of $\alpha = (2.9 \pm 0.9) \times 10^{-4}$ cm^2 s^{-1}, which yields a characteristic diffusion time $\tau = 5.5$ (+2.5/−1.3) s.

** The functions erf(x) and erfc(x) are defined as (Kreyszig 1999): $\mathrm{erf}(x) = (2/\sqrt{\pi}) \int_0^x \exp(-w^2)\, dw$ and $\mathrm{erfc}(x) = 1 - \mathrm{erf}(x)$.

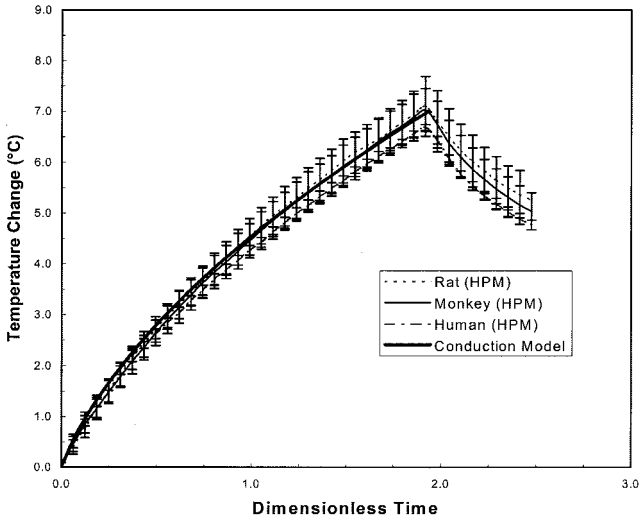

Fig. 4. Skin surface temperature increases for the HPM exposures are compared with the conduction model. Temperatures are plotted against dimensionless time ($t\alpha L^{-2}$).

Low-power exposures

The results of the low-power exposures (180 s at 175 mW cm^{-2}) for the three species are presented as Fig. 5. There is a greater range of temperature increase response and greater variability of responses compared with the short-term, high-power exposures.

The monkey, rat, and human mean skin temperature increases coincide during the initial portion of the heating curve. Approximately half way through the 3-min exposure, the human results plateau at approximately 8.3°C temperature increase; the monkey and rat data do not exhibit this phenomenon but continue to increase during the exposures. Over the course of the exposure, the rat mean skin surface temperature increases approximately 2°C more than does the human mean skin surface.

The low-power temperature data are compared with the model predictions in Fig. 6. Similar to Fig. 4, the time variable is non-dimensionalized by the estimated thermal diffusion time, $\tau = 1.6$ s. As the parameter σ represents a dimensionless power density, the value of $\sigma = -0.53$ is based on the best fit to the rat data for the high-power exposures ($\sigma = -3.01$), adjusted proportionally for the power density (175 mW cm^{-2}/1,000 mW cm^{-2}). Three different skin blood flow rates are depicted in Fig. 6: low blood flow (LBF; $\Omega = 0.0015$), moderate blood flow (MBF; $\Omega = 0.005$), and high blood flow (HBF; $\Omega = 0.010$). These correspond, respectively, to approximate skin blood flow rates of 5 mL (100 g)$^{-1}$ min^{-1}, 17 mL (100 g)$^{-1}$ min^{-1}, and 33 mL (100 g)$^{-1}$ min^{-1}.

DISCUSSION

Historically, RF exposure standards have been based on limiting the specific absorption rate. Maximum permissible exposure levels are based on a nominal safe exposure criterion SAR ≤ 0.4 W kg^{-1} in the frequency range 0.1 MHz $< f <$ 6.0 GHz. At higher frequencies (which include the MMW band), ". . . the exposures are quasi-optical, and . . . power density is the meaningful parameter" (IEEE 1999). Thus, the use of either whole-body or local specific absorption rate to establish safe exposure levels is not appropriate to this frequency band.

The Institute of Electrical and Electronics Engineers (IEEE) standards for human exposure to RF fields specify maximum permissible exposure (MPE) levels for

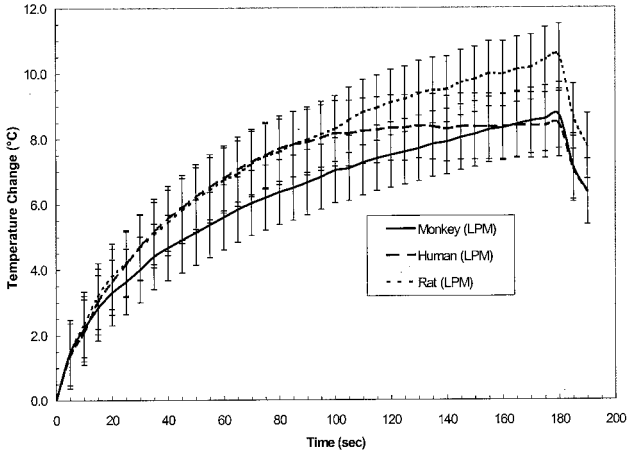

Fig. 5. The mean temperature increases are shown for 180-s LPM exposures of the three species. Four subjects per species were exposed, with three repeated exposures per subject. Error bars represent ±1 S.D. of the species ensemble.

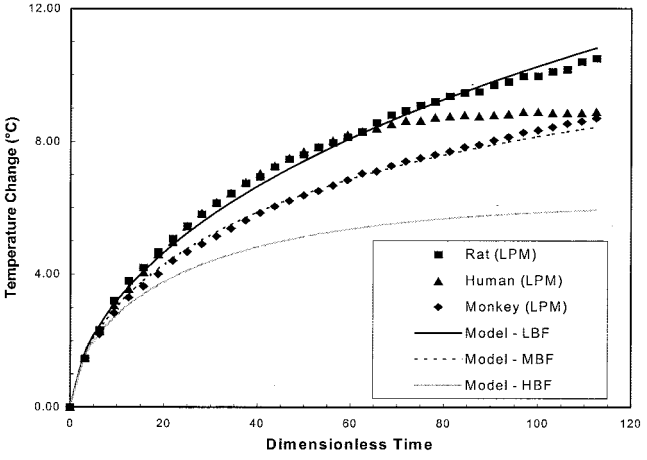

Fig. 6. Skin surface temperature increases are shown for the HPM exposures and are compared with model predictions for three different skin blood flow rates: Low Blood Flow (LBF), Moderate Blood Flow (MBF) and High Blood Flow (HBF). Temperatures are plotted against dimensionless time ($t\alpha L^{-2}$).

MMW irradiation. For MMW exposures in controlled environments, the whole-body MPE in terms of power density is 10 mW cm^{-2}, based on an averaging time of between 10 s and 6 min (depending on frequency). For partial-body exposures, the standard allows a local time-averaged power density as high as 40 mW cm^{-2} over similar averaging times (IEEE 1999).

The exposure guidelines are based, at least in part, on experiments on laboratory animals (IEEE 1999). In extrapolating estimates of human skin surface temperature increases—as functions of power density, exposure time, and wavelength—from animal experiments, it is critical to understand thermoregulatory differences between the species and the roles those mechanisms play in determining the thermal response to MMW irradiation.

High-power exposures

The results of the short-term, high-power exposures (Fig. 3) show the surface temperature increase responses for the three species are indistinguishable from each other.

For short-term exposures (duration less than 10 s), there may not be time for a thermoregulatory response (vasodilation) to be expressed. Under those conditions, a resting subject would not be expected to show any effects from changes in skin blood flow, and a conduction model is appropriate. Walters et al. (2000) demonstrated the one-dimensional diffusion model can be applied to predict surface temperatures for short-term, high-power MMW exposures on the human back. Under these conditions (high power density, short exposure duration) the temperature response may be described by a one-dimensional conduction model that neglects blood flow and surface cooling.

The one-dimensional conduction model can be successfully employed to describe surface temperature increases at locations other than the back, and for a variety of species (Fig. 4). The similarity of responses across species validates the assumption the tissue properties (permittivity, thermal conductivity) are similar for the three species.

Low-power exposures

Longer-term exposures, such as those producing the results depicted in Fig. 5, may invoke thermal responses that include surface vasodilation and sweating in humans. This indicates caution must be used in applying results across species. Rhesus monkeys sweat, though not as efficiently as humans. Rats do not sweat, although conscious rats can induce localized evaporative cooling by licking the fur. Humans are unique in that their primary thermoregulatory responses to heat stress are "... active vasodilation of the skin arterioles and increased sweating" (Rowell 1986). Sweating that commences prior to the start of exposure does not appear to significantly depress the temperature increase but does lower the baseline skin temperature, thereby reducing the maximum skin temperature achieved (Nelson et al. 2000).

Assuming an initial temperature difference $\theta = -4°C$, the three skin blood flow rates depicted in Fig. 6 (LBF, MBF, HBF) approximately correspond to 5 mL 100 g^{-1} min^{-1}, 17 mL 100 g^{-1} min^{-1}, and 33 mL 100 g^{-1} min^{-1}, respectively. Although these values are hypothetical, they are within physiologic range. The lowest blood flow value (5 mL 100 g^{-1} min^{-1}) is consistent with skin blood flow rates in the arms of anesthetized baboons (Hales et al. 1979) and in humans (Charny et al. 1987) under normothermic conditions.

The highest blood flow rate shown (33 mL 100 g^{-1} min^{-1}) characterizes average (total) blood flow under local, mild heating conditions. Johnson et al. (1976) measured a skin blood flow rate of approximately 30 mL 100 g^{-1} min^{-1} in the forearm of a human subject in which the (forearm) skin was heated to 42.5°C. Song et al. (1980) measured a skin blood flow rate of approximately 29 mL 100 g^{-1} min^{-1} in the legs of anesthetized rats warmed in a bath at 43°C for 1 h.

Substantially higher blood flow rates may be achieved under more severe heating conditions. Rowell (1986) estimates a skin blood flow rate in the human forearm of 217 mL 100 g^{-1} min^{-1} during whole-body heating. As maximal vasodilation response can be induced with localized skin heating only (Taylor et al. 1984), similar flow rates might be achievable by heating the human forearm only.

The low blood flow rate model (LBF, Fig. 6) describes the rat skin temperature increase. This is also consistent with the human forearm temperature increase for the initial two-thirds of the long-term exposure while a moderate blood flow rate (MBF, Fig. 6) replicates the monkey forearm data. The onset of vasodilation may explain—at least in part—the plateau effect seen in human subjects at approximately 90 s exposure time (Fig. 5).

These results are consistent with a vasodilation thermoregulatory response occurring at approximate exposure time $t = 90$ s. The fact this behavior is not observed in either the monkey or the rat is consistent with the minimal vasodilation response in those species (compared with the human) for the temperature ranges experienced. Current studies are underway to determine the onset of the vasodilation response through laser doppler imaging (LDI) of the skin surface.

Some of the inter-species variations in surface temperature response may be attributable to differences in the experimental protocol—specifically, the anesthesia employed for the monkey and rat experiments. This could affect the thermoregulatory responses of those species and account for some differences in the skin temperatures when compared with the results obtained from the human exposures.

Longer-term exposures may entail conduction into the sub-dermal tissues. This would suggest possible inter-species, intra-species and intra-subject variability in skin temperature response due to differences in epidermal and dermal tissue thicknesses. Long-term, low-power exposure of other surface regions may result in differing temperature responses, due to variations in tissue layer thicknesses, adiposity, blood flow, or other factors. Gender might also affect response to prolonged surface heating.

Comparison with MPE

The IEEE Standard C95.1 states a maximum permissible exposure (MPE) of 10 mW cm^{-2} for whole-body MMW exposure in both controlled and uncontrolled environments. For partial-body exposures (excluding the eyes and testes), the MPE may be as high as 40 mW cm^{-2}, depending on frequency (IEEE 1999). Under conditions of low cutaneous blood flow (5 mL 100 g^{-1} min^{-1}), eqn (5) predicts a skin surface temperature increase of 2.0°C for a 180-s exposure at the MPE (40 mW cm^{-2}). Note this is a conservative estimate, as it assumes no thermoregulatory response (i.e., no increased blood flow) and no heat loss to ambient.

As the threshold temperature for skin damage (hyperemia, without epidermal loss) is approximately 45°C (Ryan et al. 2000), it is unlikely irradiation at the MPE would produce thermal injury, at least for exposures up to three minutes duration.

CONCLUSION

Surface temperature increases from short-term, high power density exposures can be described by a one-dimensional conduction model that neglects blood flow effects and surface cooling. There is little or no difference in skin surface temperature response among the three species for the anatomical sites considered (rat abdomen, human and monkey forelimbs).

Skin surface temperature response from sustained, low-power exposures vary between species for the anatomical sites considered. In the human forearm, the temperature response may be affected by the onset of vasodilation. Caution must be exerted in inter-species extrapolation of surface heating results from sustained, low-power MMW irradiation, as results can be affected by cutaneous blood flow and the magnitude of vasodilatation response.

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REFERENCES

Arpaci VS. Conduction heat transfer. Reading, MA: Addison-Wesley; 1966.

Blick DW, Adair ER, Hurt WD, Sherry CJ, Walters TJ, Merritt JH. Thresholds of microwave-evoked warmth sensations in human skin. Bioelectromagnetics 18:403–409; 1997.

Carl JR, Arndt GD, Fink PW, Beer NR, Henry PD, Pacifico A, Raffoul GW, inventors. The United States of America, assignee. Endothelium preserving microwave treatment for atherosclerosis. U. S. patent 6,047,216. 2000 April 4.

Charny CK, Hagmann MJ, Levin RL. A whole body thermal model of man during hyperthermia. IEEE Trans Biomed Eng BME-34:375–387; 1987.

Durney CH, Massoudi H, Iskander MF. Radiofrequency radiation dosimetry handbook. Brooks AFB, TX: USAF School of Aerospace Medicine, Aerospace Medical Division; 1986.

Foster KR, Kritikos HN, Schwan HP. Effect of surface cooling and blood flow on the microwave heating of tissue. IEEE Trans Biomed Eng 25:313–316; 1978.

Gandhi OP, Riazi A. Absorption of millimeter waves by human beings and its biological implications. IEEE Trans Biomed Eng MTT-34:228–235; 1986.

Gilbert RK, Zoratti PK, Becker R, Brumbaugh G, Chaplin T, Harrison M, Hawks M, Gondoly K. Characterization and evaluation of a prototype forward-looking automotive radar (FLAR) final report. Ann Arbor, MI: Environmental Research Institute of Michigan; Report DTNH22-94-Y-17016; 1997.

Hales JRS, Rowell LB, King RB. Regional distribution of blood flow in awake heat-stressed baboons. Am J Physiol 237:H705–H712; 1979.

Institute of Electrical and Electronic Engineers. IEEE standard for safety levels with respect to human exposure to radio frequency electromagnetic fields, 3 kHz to 300 GHz. New York: IEEE; IEEE Standard C95.1; 1999.

Johnson GS, Elizondo RS. Thermoregulation in macaca mulatta: a thermal balance study. J Appl Physiol 46:268–277; 1979.

Johnson JM, Brengelmann GL, Rowell LB. Interactions between local and reflex influences on human forearm skin blood flow. J Appl Physiol 41:826–831; 1976.

Kolka MA, Elizondo RS. Thermoregulation in erythrocebus patas: a thermal balance study. J Appl Physiol 55:1603–1608; 1983.

Kreyszig E. Advanced engineering mathematics. New York: John Wiley & Sons; 1999.

Mitchell JW, Galvez TL, Hengle J, Myers GE, Siebecker KL. Thermal response of human legs during cooling. J Appl Physiol 29:859–865; 1970.

Moffa P, Austin J, Dow GS, Ikizyan I, Hibben M. Development of performance specifications for collision avoidance systems for lane change, merging, and backing—task 5: crash countermeasure technology investigation. Washington, DC: U.S. Department of Transportation, National Highway Traffic Safety Administration; Report DOT HS 808 506; 1996.

Nelson DA, Nelson MT, Walters TJ, Mason PA. Skin heating effects of millimeter-wave irradiation—thermal modeling results. IEEE Trans Microwave Theory Tech 48:2111–2120; 2000.

Nikawa N, Hoshi N, Kawai K, Shigeyuki E. Study on dental diagnosis and treatment using millimeter waves. IEEE Trans Microwave Theory Tech 48:1789–1799; 2000.

Pakhomov AG, Akyel Y, Pakhomova ON, Stuck BE, Murphy MR. Current state and implications of research on biological effects of millimeter waves. Bioelectromagnetics 19:393–413; 1998.

Pennes HH. Analysis of tissue and arterial blood temperatures in the resting human forearm. J Appl Physiol 1:93–122; 1948.

Rowell LB. Human circulation regulation during physical stress. New York: Oxford University Press; 1986.

Ryan KL, D'Andrea JA, Jauchem JR, Mason PA. Radio frequency radiation of millimeter wave length: potential occupational safety issues related to surface heating. Health Phys 78:170–181; 2000.

Shelton WW. Millimeter-wave exposure system augmentation. Brooks Air Force Base, TX: Armstrong Laboratory Technical Report (AL-TR-1991-0056); 1991.

Song CW, Rhee JG, Levitt SH. Blood flow in normal tissues and tumors during hyperthermia. J Natl Cancer Inst 64:119–124; 1980.

Stitt JT, Hardy JD. Thermoregulation in the squirrel monkey (saimiri sciureus). J Appl Physiol 31:48–54; 1971.

Taylor WF, Johnson JM, O'Leary D, Park MK. Effect of high local temperature on reflex cutaneous vasodilation. J Appl Physiol 57:191–196; 1984.

Walters TJ, Blick DW, Johnson LR, Adair ER, Foster KR. Heating and pain sensation produced in human skin by millimeter waves: comparison to a simple thermal model. Health Phys 78:259–267; 2000.

Werner U, Giese K, Sennhenn B, Plamann K, Kolmel K. Measurement of the thermal diffusivity of human epidermis by studying thermal wave propagation. Physics Med Biol 37:21–35; 1992.

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